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Health Hazards of Combustion Products From Aircraft Composite Materials

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Sanjeev Gandhi* and Richard E. Lyon	

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16. Abstract

Concerns about the potential health hazards of burning fiber-reinforced polymer composites in aircraft fires parallel the rising usage of these materials for commercial aircraft primary and secondary structures. An overview of the nature and the potential hazards associated with airborne carbon fibers released during flaming combustion of aircraft composites is presented. The current data derived from animal studies are insufficient to determine the acute toxicity of carbon fibers from burning composites. Further work is needed to examine the adverse health effects of volatile organic chemicals and to assess if any synergistic interactions exist with the fibers.

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EXECUTIVE SUMMARY

Fiber-reinforced polymer composites are increasingly being used for primary and secondary structural components in aircraft. Incineration of composites in postcrash aircraft fires generates a complex mixture of combustion products comprised of gases, organic vapors, and particulate matter including fibers. There are concerns regarding the health hazards posed to fire fighting, investigation, and cleanup personnel exposed to these combustion products. This report presents a literature review on the potential health hazards due to inhalation of carbon fibers and associated organic compounds released from composites in aircraft fires. The toxicology of fibers is reviewed and animal studies on inhalation toxicity of carbon fibers are included. Data derived from earlier studies on the production and physical characterization of fibers are presented.

The fiber dimensions and total deposition deep into the lung determine the inhalation hazard from fibers. Fibers with diameters smaller than 3 μm and lengths shorter than 80 μm are respirable and can penetrate deep into the lungs. The fiber retention time inside the lung is primarily dependent on the fiber dimensions. Fibers smaller than 15 μm long are cleared from the lungs by cellular activity. However, longer fibers saturate the self-clearance mechanism of the lungs and can lead to pathological effects. Animal studies on exposure to respirable size raw polyacrylonitrile (PAN) and pitch-based carbon fibers do not indicate that there are significant adverse health effects. Studies involving animals exposed to aerosols of composite dust and carbon fibers from machining and grinding of fiber composites are inconclusive with respect to pathological effects.

Data from fire tests and crash-site investigations suggest that only a small fraction of the fibers released in fires is of respirable size. However, detailed chemical analysis of organic vapors revealed a high number of toxic organic compounds that are associated with the fibers. Several of the organic chemicals are known carcinogens and mutagens in animals. Further work is needed to assess the health implications of any synergistic interactions between the chemical and fibrous combustion products.

INTRODUCTION

Aircraft accidents involving fire and explosion of fiber-reinforced composite materials present unique environmental, safety, and health hazards. The three basic health hazards from fiber-reinforced polymer composites in a crash-fire situation are the sharp splinters of exposed material, the fibrous dust dispersed by the fire or blast, and the toxic gases generated during burning of the polymer matrix resin in the fire. With the dramatic increase in fiber composites in primary and secondary structural components of new commercial transport aircraft, there is growing concern among the aircraft safety and mishap response and management community about the health hazards of exposure to airborne carbon fibers released in a postcrash fire.

The Boeing 777 shown in figure 1 illustrates the level of polymer composite usage in current generation transport category aircraft. The vertical and horizontal tail sections as well as major wing sections are now made of toughened carbon fiber-reinforced epoxy composite. [1]

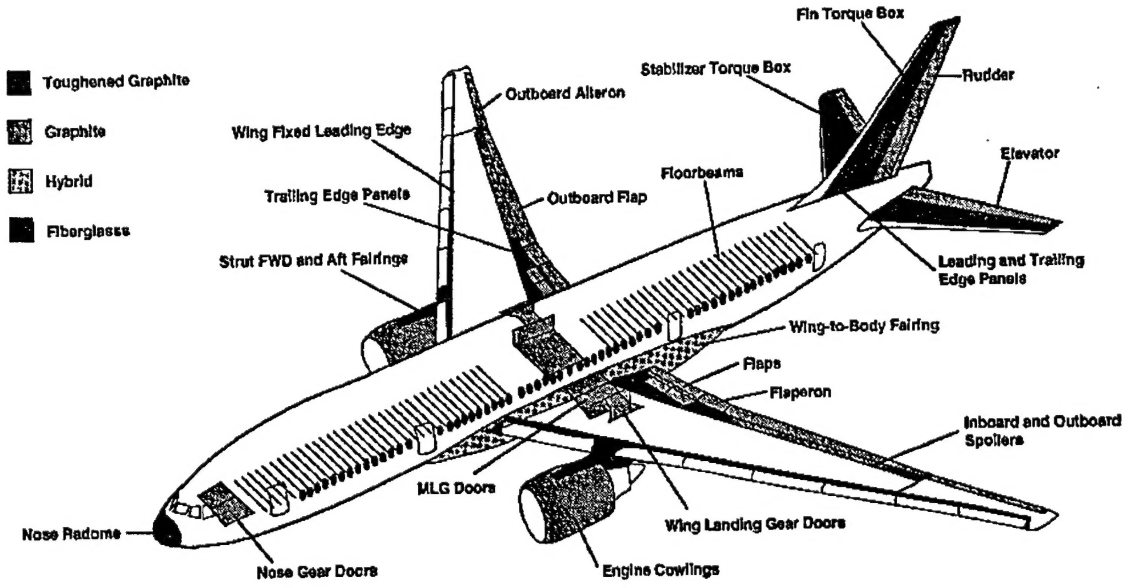

FIGURE 1. ADVANCED COMPOSITES ON BOEING 777

Response personnel are exposed to hazards posed by the fibrous particulates following a crash and the subsequent fire from spilled fuel. In recent years, a number of incidents have been reported on the irritant and toxic effects of fibrous matter and aerosols on personnel responding to the crash site. [2-4] Firefighters and emergency crews involved in postcrash cleanup and restoration operations have expressed concerns about long-term effects from exposure to carbon fibers released from burning composites and in the special needs for extinguishing and handling the incinerated fiber composites. [3-6] Incident reports vary concerning the nature and severity of short- and long-term adverse effects on the responding crews. Rescue and recovery personnel have suffered varying degrees of adverse health effects, ranging from eye and skin irritation to severe respiratory problems. In certain instances, response teams equipped with enhanced protective clothing reportedly suffered from penetration by strands of needle-sharp carbon fibers [4]. Reported effects of a chronic nature include forced respiration volume, reduced

exercise capability, and a positive histamine challenge test. [7] Histamine is a chemical present in the body tissue; when released it may cause allergic reactions that lead to respiratory problems due to the constriction of bronchial capillaries. A definition of the term is provided in the glossary.

The potential health risks associated with fires involving fiber-reinforced polymer composites include the fibrous dust and airborne particles released during burning that can be inhaled and deposited in the deep lung region and the sharp fiber fragments that can penetrate the skin. As the organic resin burns off, the continuous, reinforcing fibers are exposed to a turbulent, oxidizing environment that causes them to break up and erode into small microfiber fragments. The concentration of respirable airborne microfibers increases when the accident involves fire and impact or explosion. In addition, the toxic chemicals produced from the combustion of the organic resin may be adsorbed on respirable fibers and enter the respiratory system with acute or chronic effects.

Another aspect of the potential risk following an aircraft accident is the short-circuiting of electrical and electronic systems due to the conductive nature of carbon fibers. The airborne fibers released from burning composites are carried by the fire plume and dispersed downwind in the atmosphere. Pitch- and polyacrylonitrile (PAN) -based carbon fibers have electrical resistivities of about 250 and 1800 microhm-cm, respectively, which is on the order of 10 percent of the conductivity of metals. Such a scenario poses potential risk to nearby power distribution lines, transformers, radar, and electronic equipment.

To date combustion toxicology has focused on the acute and chronic effects of gases produced by the incomplete combustion of flammable materials inside a compartment. Vapors and gases vitiate a compartment and at high concentrations cause fatal or incapacitating effects among occupants. As a result, a number of bench-scale test methods and analytical methodologies have been developed for evaluating the toxic potency of the combustion products. [8-9] Most test methods involve analytical testing for a select group of gases and animal studies for inhalation toxicology. [9] In a recent article, Hall [10] chronicled the evolution of various approaches to the evaluation of combustion toxicity. Purser [11], Hartzell [12], and Speitel [13] have presented detailed reviews of the advances in the measurement of smoke toxicity over the past 3 decades. In contrast, toxicity of aerosols given off in a fire, i.e., the solid component of smoke such as soot, condensates, and fibers, has received little attention. Some studies have focussed on the toxicity of soot particles since these are the most common particulate components of smoke [14, 15]. Soot is formed through aggregation of unburned hydrocarbon resulting from incomplete combustion of organic materials. The toxicity of soot depends primarily upon the type and amount of chemical species adsorbed on the surface. Henderson reviewed animal studies on the chronic and acute toxicity of diesel engine exhaust comprised of low aspect (length-to-diameter) ratio particulate matter. [14] There is, however, a paucity of information on the short- and long-term health effects of the high aspect ratio, micron-sized fibers that become airborne during the burning of fiber-reinforced polymer composites.

This report presents a review of the nature of hazards posed by particulate matter, fibers and associated chemicals, released from burning composites. In view of the current and projected use of composites in commercial aircraft, these hazards are of great concern to fire and rescue

personnel, investigators, and cleanup and recovery crews at the aircraft crash site. Characteristics of fibers released from burning composites and their size distribution are described. An overview of several animal studies to assess the inhalation toxicity of fibers is presented.

BACKGROUND

Fiber-reinforced polymer composites are lightweight functional substitutes for metals in civilian and military aircraft with a superior strength-to-weight ratio and corrosion resistance. Fiber composite structures are 25-45 percent lighter than their metallic counterparts. The higher initial fabrication and maintenance costs of polymer composites compared to metals are offset by fuel savings over the lifetime of the aircraft or by enhanced performance. The processing technology and design database for fiber-reinforced composites has matured to the point where these materials are now being used extensively for primary and secondary structures in commercial transport and commuter aircraft. [16]

Lower fuel costs brought about by lighter-weight structural components will lead to major improvements in the life-cycle performance of the aircraft. To achieve improved life-cycle performance, manufacturers are exploring increased use of lightweight structural composites in next-generation commercial aircraft. [16, 17] Figure 2 shows the steady increase in the use of fiber-reinforced composite components since 1981 by the two biggest suppliers of civil transport airplanes. The percent structural weight is based on the operating empty weight of the aircraft.

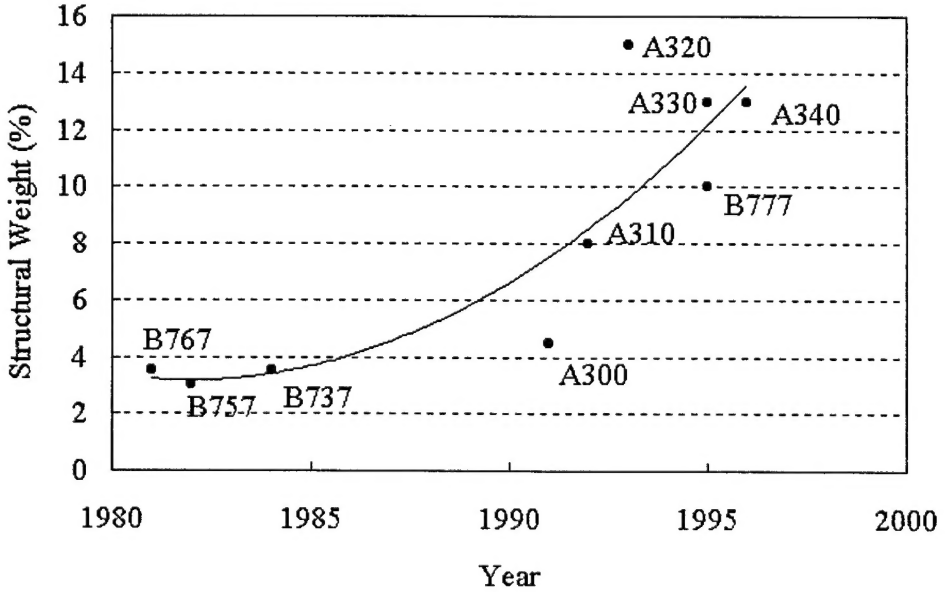

FIGURE 2. STRUCTURAL WEIGHT (PERCENT) OF FIBER COMPOSITES IN BOEING (BXXX) AND AIRBUS (AXXX) COMMERCIAL TRANSPORTS

It is apparent that the Airbus family of products has led the aircraft industry in the use of fiber composite structures. The Boeing 777 aircraft with a three-fold increase in the structural weight of composite parts, compared to the previous generation airplanes, represents a significant advance. Boeing estimates that the average structural weight fraction of polymer composites in

their commercial airplanes will increase from about 7 percent currently to about 20 percent over the next 15 years. [18]

COMPOSITION OF POLYMER COMPOSITES

Polymer structural composites are engineered materials comprised of continuous, high-strength fibers impregnated with a polymer matrix to form a reinforced layer (ply), which is subsequently bonded together with other layers under heat and pressure to form an orthotropic laminate. The strength and stiffness of the laminate are determined by the orientation of the fibers with respect to the loading direction and their volume fraction in the composite. For a typical polymer matrix composite, fibers comprise about 55-60 volume percent of the laminate with the polymer resin being the remainder.

Fibers used for composite reinforcements, in order of increasing cost (decreasing quantity), are high-strength glass, aramid, carbon/graphite, and boron. E-glass is the predominant reinforcement for polymer matrix composites used in aircraft interior applications due to its low moisture affinity and superior electrical insulating and mechanical properties. Other commercial compositions of glass fiber include S- and R-glass, which offer improved modulus with higher strength and heat and greater chemical resistance respectively.

Carbon and boron fibers are premium priced, high-performance reinforcement materials used in military and commercial aircraft primary structures. Continuous carbon fibers are the preferred material for reinforcement in applications where a significant reduction in weight is needed along with improved functional performance. Carbon fibers have density ρ = 1850 kg/m^3, axial Young's modulus of elasticity E = 200-400 GPa, and tensile strength σ = 2-7 GPa. These numbers compare to ρ = 7900 kg/m^3, E = 200 GPa, and σ = 0.7 GPa for steel; and ρ = 2700 kg/m^3, E = 69 GPa, and σ = 0.3 GPa for aluminum alloys. Consequently, the stiffness and strength per unit weight (specific stiffness and strength) of carbon fibers, obtained by dividing the Young's modulus and tensile strength by the density of the material, is about 6 and 30 times the specific strength and stiffness, respectively, of steel and aluminum. For this reason alone, carbon fibers are making inroads into weight-critical applications such as aerospace.

Fibers are used alone or in combination with other fibers (hybrids) in the form of continuous fiber fabrics, tapes, and tows or as discontinuous chopped strands. Carbon and graphite fibers are high-carbon materials manufactured in a continuous process from precursor fibers, polyacrylonitrile (PAN) and pitch. The process involves controlled pyrolysis at 1000 to 3000°C of the precursor fiber to produce carbon and graphite fibers containing 93-95 and 99 percent carbon atoms, respectively. [17] Typical carbon fibers used in composite manufacture are 6-8 μm in diameter.

Boron fibers consist of a tungsten core onto which boron trichloride is vapor-deposited forming a high-strength strand 100-150 μm in diameter. Borsic filament is another form of boron material that consists of a boron filament coated with silicon carbide. [19]

The two main classes of polymer resins used as the fiber matrix in composites are thermosets and thermoplastics. Epoxy resins are the most common thermoset resins in commercial aircraft

applications because they are relatively tough, easy to process, and have moderate temperature capability. Unfortunately, epoxies are very flammable and cannot be used as matrix materials in composites for aircraft interior components that must pass strict heat release rate regulations. Phenolics are the thermoset resin of choice for aircraft interiors solely because of their low heat release rate. Other thermosetting polymers in order of decreasing temperature capability include cyanate esters, bismaleimides, polyesters, and vinylesters—all of which have high heat release rates when burned.

Thermoplastic resins have found limited use as matrix resins in aircraft interior and structural composites because they require high forming temperatures. Thermally stable engineering thermoplastics, such as polyetheretherketone and polysulfone, are used without fiber reinforcement for molded parts in cabin interiors and as thermoset resin tougheners in commercial and military applications. [18] The development of high-performance thermoplastic resin systems is an area of evolving research that holds great promise for future applications of polymer matrix composites.

CARBON FIBER TOXICOLOGY

Extensive studies have been accomplished regarding health hazards associated with natural and man-made mineral fibers such as asbestos and glass. The existing framework of information on asbestos and glass fibers delineates the contributing factors in fiber toxicity, which may be applied for consideration of carbon fiber toxicology. Warheit [20] reviewed the toxicity of asbestos, rock wool, fiberglass, and organic fibers like aramid and carbon. Asbestos, a naturally occurring mineral, is the most widely studied fiber for human health effects. Inhalation toxicity of asbestos is primarily attributed to the fiber's dimensions. [20] Other comprehensive reviews on the health assessment of natural and man-made fibers can be found in references 21-24. Vu [22] and Hesterberg [24] particularly described the health risks from chronic, long-term exposure in the work environment. These studies do not address the health hazards of a large single-dose (acute) exposure due to gaseous and fibrous matter emitted during combustion of composites.

There are two major routes to exposure from fibers—dermal and inhalation. Irritation of the skin and eyes is a typical response to dermal exposure resulting from a reaction to sharp, fragmented fibers of diameter greater than 4-5 μm. The severity of the exposure depends on the fiber size and stiffness and the irritation is not permanent.

The inhalation hazard from fibers poses the greatest potential for adverse health effects on humans and depends on the total dosage and the physical dimensions of the fibers. Smaller particles are deposited in the lower regions of the lungs and chronic toxicity is associated with this type of exposure. For purposes of hazard assessment, fibers are defined as high aspect ratio particles having a length-to-diameter ratio (L/D) greater than three. [20] The criterion L/D >3 has been established as an essential condition for chronic toxicity in studies of natural and man-made mineral fibers. [23]

Numerous studies indicate that exposure to asbestos can cause pulmonary fibrosis, lung cancer, and mesothelioma, a cancer of the pleural cells lining the lungs. The toxic effects of fibers are a

consequence of their biophysical properties (dimensions and time of deposition) and the total ingested dose of fibers. Warheit [21] has summarized the unique toxicological effects induced by fibers. An important distinction can be made between fibers and chemicals when assessing the dose-response relationship. In the case of chemicals, the agent itself changes due to metabolic reactions within body fluids and it is excreted from the body after producing the pathological response. [25] In contrast, fibers are inert and can be long lasting.

The dose (amount) of fibers deposited in the pulmonary region depends on the fiber density, size, and shape. Fibers entering the respiratory tract are restricted to those with dimensions capable of penetrating the tracheobronchial path after traversing through the nose and the upper respiratory tract. [26-27] The respirability of a fiber is characterized in terms of the aerodynamic diameter D_a, which refers to the diameter of an equivalent spherical particle having the same terminal velocity as the fiber. Fibers with D_a <10 μm are most likely to penetrate into the gas exchange region of the pulmonary system. Fibers with diameters smaller than 3 μm and lengths <80 μm fall in the respirable range and can penetrate deep into the lungs. [7, 20]

Figure 3 illustrates the five modes of fiber deposition in the bronchial airways. The airways region in the human lung consists of a series of branching capillaries called bronchioles that become progressively smaller. The multiple division of the bronchi greatly increases the total cross-sectional area of the airways available for fiber deposition. [27] The aerodynamic diameter governs the mechanism of fiber deposition in the airways. In larger airways, fibers with greater terminal velocity are deposited by impaction against the inside walls of airways. Impaction against the airway walls occurs where the fibers cannot overcome their inertial drag and cannot adjust to changes in the angle and velocity of the airflow. In smaller airways where the airflow velocity becomes very small, sedimentation is the primary mode of fiber deposition. Lippman [25] has described three other mechanisms of fiber loading in lungs—interception, diffusion, and electrostatic deposition.

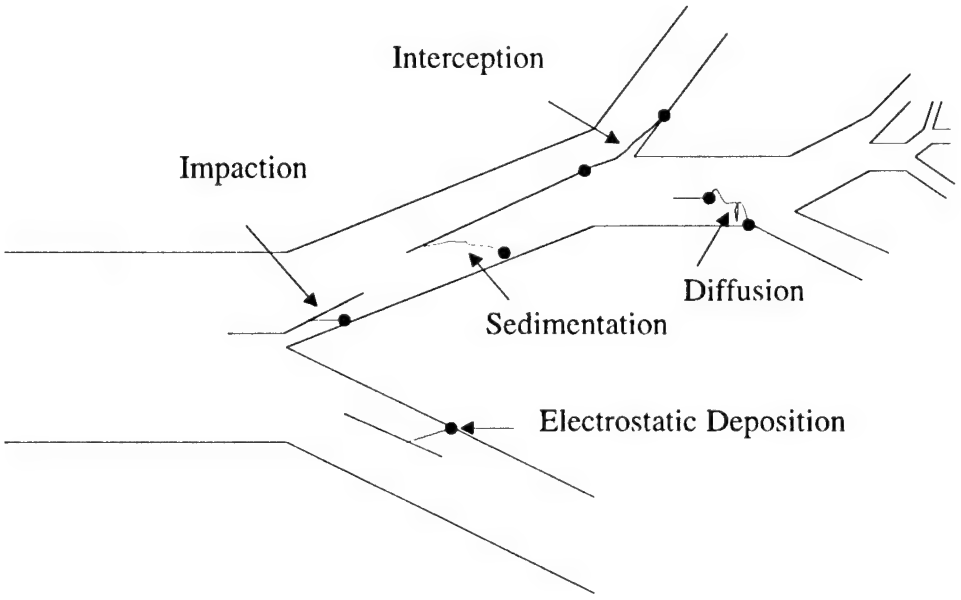

FIGURE 3. MODES OF FIBER DEPOSITION IN LUNG AIRWAYS

Fibers aligned vertical to the airway flow stream are primarily deposited by interception at each successive bifurcation. The probability of interception increases when the fiber length is greater than 10 μm. The larger airway bifurcations are the primary sites of fiber deposition and lung cancer in humans. [20] There is a lack of similar evidence for fiber deposition in the alveoli. However, animal studies have shown that fibers that are small enough to pass through the larger pulmonary airways are found selectively in the alveoli tissue at the junction of terminal bronchiole-alveolar duct. [21, 25] Warheit [20] found that the fiber deposition patterns in various rodent species, through scanning electron microscopy (SEM) analysis of lung sections, are independent of the fiber nature and shape.

Fiber retention time in the lung depends on the number of fibers, their dimensions, and the deposition site. There are two modes of fiber clearance from the lung, translocation and dissolution in lung fluids. [20] Translocation is the migration of intact fibers from their initial deposition site in the lung to other organ sites. The alveolus is composed of thin walls of epithelial cells that are very sensitive to foreign matter. Also present are pulmonary alveolar macrophages. These cells perform an important function called phagocytosis. A pulmonary self-defense mechanism, phagocytosis involves removal of bacteria, dead cells, and foreign particles and fibers by ingestion. [21, 28] It has been suggested that particles after ingestion by the alveolar macrophages are cleared through the lung's peripheral mucociliary drainage pathways. [20] Hesterberg [24] has given a detailed description of the cellular activity involving macrophage induced clearance of fibers and particulates.

The removal efficiency is limited by the macrophage diameter (~10-15 μm). Fibers with length <10 μm are easily cleared by the macrophages. However, macrophage cells cannot completely phagocytize the longer fibers (~20-30 μm). The incomplete ingestion of long fibers results in overloading of alveolar macrophages and the whole fiber clearing process is slowed down. The impairment of phagocytosis due to partial ingestion kills the macrophage cells, releasing a mixture of protein-based chemicals into the surrounding lung tissues. Morgan et al., from work in reference 21, introduced a suspension of glass fibers with an average diameter of 1.5 μm and random lengths in rat lungs and monitored fiber retention over a 2-year period. It was found that fiber retention was length dependent. After 1 year, there was only 10 percent retention for fibers 5 μm and smaller, and 20 percent retention for fibers 10-μm long. The authors also reported evidence of fiber disintegration and dissolution.

Penetration of fibers in the lung region initiates an inflammatory response called alveolitis. The lung's efforts to repair this damage manifests as progressive scarring in the lung walls. This interstitial scarring effect caused by the deposition of fibrous matter is termed pulmonary fibrosis, a condition in which scar tissue forms in the connective tissues that support the alveoli in the lungs. Scarring can be a reaction to a large number of diseases and conditions that lead to acute to chronic inflammation of lung tissues. The induction of fibrosis retards the process of fiber clearance resulting in longer fiber retention. This condition when caused by inhalation of minute asbestos fibers composed of calcium and magnesium silicate is referred to as asbestosis. Extended chronic exposure to asbestos is known to result in bronchogenic carcinoma or lung cancer. Epidemiological studies have demonstrated the causal relationship between asbestos exposure over a period of several years and development of lung cancer and mesothelioma. [25, 27]

The health hazards caused by inhalation of asbestos are related to critical fiber dimensions, that is fibers with certain critical diameters and lengths. Table 1 shows the critical fiber dimensions related to asbestosis, lung cancer, and mesothelioma. For asbestosis, the retention is highest when fiber length is in the range of 2-5 μm and the diameter is between 0.2-2 μm. The upper limit for the fiber diameter is 3 μm. The risk of lung cancer increases with the presence of a substantial number of long fibers (>10 μm). The critical limits of fiber diameter are 0.3 and 0.8 μm. It is believed that longer fibers (>10 μm) have more impact on malignancy of the lung due to their longer retention time. Shorter fibers, i.e., <5 μm are more easily cleared from the lung due to ingestion by the macrophages and by dissolution in lung fluids. [25]

TABLE 1. CRITICAL FIBER DIMENSIONS FOR ASBESTOS INDUCED DISEASES

Disease	Diameter (μm)	Length (μm)
Asbestosis	0.2-2	2-5
Lung cancer	0.3	>10
Mesothelioma	<0.1	>5

Asbestos fibers have been implicated as a cause for mesothelioma–cancer of the pleural cells due to cumulative, long-term exposure over 20-40 years. [25] These cells form the serous membrane enveloping the lungs. There are 1500-2000 cases of mesothelioma in the United States each year, mostly associated with occupational exposure with a latency period of 20 to 40 years. [21] This type of tumor originates as microscopic nodules that grow in size and diffuse through the pleural walls, invading the chest wall and the lung. The critical fiber dimensions for mesothelioma are a diameter <0.1 μm and a length of 5-10 μm. The thinner fibers can reach deep inside the alveoli due to their aerodynamic characteristics and are transported to the pleural surfaces through penetration of epithelial cell wall. [20, 25]

The fibrous particulates can also carry with them a diverse package of chemical species. Smaller particles having the highest surface-to-volume ratio pose the greatest danger with respect to the quantity and effect of adsorbed toxicants. It has been suggested that adsorbed toxicants may enhance the pathology of inhaled particles. [15] Lippman [25] argues that biophysical characteristics are important but insufficient indicators of fiber toxicity, and "other physical-chemical properties that impart biological potential to fibers should be investigated." For example in the case of inert fibers such as carbon, the presence of surface contaminants from the combustion environment might affect fiber retention in the lung.

The asbestos exposure levels in the general industry have been regulated by Occupational Safety and Health Administration (OSHA). The OSHA [29] standard limits the maximum permissible exposure concentration to 0.1 fibers/cm^3 for an 8-hour time-weighted average (TWA). The fiber is defined as having length >5 μm and an L/D ≥3. There are no OSHA standards for synthetic fibers such as fibrous glass and carbon. The National Institute for Occupational Safety and Health (NIOSH) [30] recommends that exposure be limited to 3 fibers/cm^3 for glass fibers having a diameter ≤3 μm and a length ≥10 μm. These guidelines have been developed based on

framework of information on asbestos and glass fibers. In consideration of the health hazards of carbon fibers, the existing guidelines for these fibers may be applied towards establishing limits on exposure to carbon fibers.

There is very little published literature on the inhalation toxicology of carbon fibers. A few studies have been done on the potential health effects related to chronic exposure from carbon fibers and dusts in the occupational environment. Two conferences [31-32] brought together experts from the aerospace and composites industries and government agencies to address the health implications of exposure to carbon fiber-reinforced composites. The primary focus of these proceedings was the hazards related to exposure from carbon fibers and dusts during the machining and handling of fiber composites. In reviewing the toxicology research on carbon fibers, Thomson [33] concluded that animal studies data indicate that there are no long-term health risks associated with exposure to PAN-based carbon fibers under occupational conditions. The health effects are limited to temporary irritations of the skin and upper respiratory tract since exposure is limited to relatively large-diameter fibers (6-8 μm).

Martin et al. [34] evaluated the potential health effects of dusts generated during the machining of six carbon fiber-epoxy composites. This was the first animal study to focus on the cytotoxicological responses caused by mechanically generated aerosol from composites rather than raw carbon fibers. Characterization of the aerosol revealed very few fibers in the generated particles; the diameter of which ranged between 7-11 μm. The mean diameter of the respirable fraction of nonfibrous particles was 2.7 μm. The particles were introduced via intratracheal injection directly into rat lungs. *In vitro* studies were also done using rabbit alveolar macrophages. The assessment of pathological damage was based on several endpoints including cytotoxicity, phagocytosis, and the presence of certain enzymes and proteins in the lavage fluid. The results indicated that four of the composite samples showed little toxicity, but two samples from carbon fiber-epoxy composites cured with an aromatic amine agent were significantly more toxic than controls. The authors suggested carbon fiber and composite dusts from these amine-containing composites should be treated with more strict exposure limits as for nuisance dusts, and further studies with longer-term inhalation exposure were needed to characterize their toxic effects. These findings are consistent with the carcinogenicity of aromatic amines used as curing agents, e.g., 4, 4' methylenedianiline (MDA). [35] The Occupational Safety and Health Administration (OSHA) lists MDA as an animal carcinogen and as a suspect human carcinogen by any exposure route: ingestion, inhalation, or dermal. [36]

The intratracheal instillation technique for assessing the toxicological response of animals involves directly injecting a known quantity of fibers into the lungs for short-term screening studies to measure fiber retention and lung defense mechanisms. In a recent workshop, [37] a panel of fiber toxicologists concluded that direct intratracheal instillation is an acceptable alternative to inhalation exposure provided that low fiber doses are used to prevent the clumping of fibers and saturation of lung cells. Henderson et al. [38] conducted a comparison of the lung response in rats subjected to inhalation exposure and intratracheal instillation of particles of known toxicity. The authors concluded that the inflammatory response in the lungs can be determined using either inhalation exposure or through direct intratracheal instillation. However, it has been suggested that the nonphysiological manner of introduction of particles in intratracheal instillation results in an irregular, nonuniform distribution of particles in the target

area. This prevents the normal fiber translocation and clearance mechanisms to play a role in determining the total fiber deposition in the airways and affects the fiber clearance mechanism involving alveolar macrophages. Warheit [39] has proposed a short-term inhalation bioassay technique using rats for the prediction of pulmonary fibrosis in humans.

Kwan [40] characterized the physical size and aerodynamic properties of particulates generated during laser machining of composites. The particle size analysis did not reveal generation of any fiber-like particles with L/D>3. The respirable fraction consisted of spherical particles with D_a <2 μm. Kwan's study did not include animal testing for toxicity. Chemical analysis of the sampled particulate matter revealed the presence of adsorbed volatile organic compounds with aromatic structure. Most of these chemicals were polynuclear aromatic hydrocarbons (PAH). The NIOSH database of toxic chemical substances describes the toxicological effects of these PAH species to range from sensory irritation to carcinogenicity. [40]

Table 2 provides a summary of the known studies on inhalation hazard of carbon fibers. In Owen's study, [41] rats were exposed to 7 μm PAN-based carbon fibers at aerosol concentration of 40 f/cm^3 for 6 hr/day, 5 days/week, for up to 4 months. The physiological responses of animals were monitored for up to 32 weeks. No pulmonary inflammation, lung function impairment, or fibrosis effects were detected. Thomson [42] tested the respirability of carbon fibers 3.5 μm in diameter and 3.5 mm long. The L/D ratio of these PAN-based carbon fibers was 1000. Rats were exposed to fiber concentration of 40, 60, and 80 f/cm^3 for 1hr/day for 9 days. No changes in physiological response were observed after post-exposure periods of 1 and 14 days. Micrographs of lung tissue did not reveal presence of carbon fibers in SEM analysis. Studies of both Owen [41] and Thomson [42] used carbon fibers with physical dimensions beyond the respirable range, i.e., fiber diameter >3 μm in the former and length much greater than 80 μm. The toxicological significance of these studies is questionable because of the nonrespirable nature of fibers used.

TABLE 2. SUMMARY OF CARBON FIBER TOXICITY STUDIES

	Fiber Parameters			Exposure Conditions			Post-Exposure Recovery	Results	Reference
Species	D (μm)	L (μm)	Conc. (f/cm^3)	hrs/ day	days/ week	weeks			
Rat	7	20-60	40	6	5	16	32 weeks	– No adverse effect on lung functions – No fibrosis	Owen [41]
Rat	3.5	3500	40-80	1	5	2	1, 14 days	– No carbon fibers in any tissue – No abnormality of pulmonary functions	Thomson [42]
Rat	3	10-60	40	6	5	16	35, 80 weeks	– Some nonfibrous particles in lung tissue – No histopathology or abnormal pulmonary functions	Waritz [43]
Rat	1-4	NA	50-90	6	5	-	4, 12 weeks	– Temporary lung inflammation, reversible after 10 days – No histopathological response or fibrosis of the lung tissue	Warheit [44]

Waritz [43] conducted an inhalation study on rats exposed to carbon fiber aerosol for 6 hours/day, 5 days a week, for 16 weeks. The carbon fibers were derived from a polyacrylic instead of a polyacrylonitrile (PAN) precursor. The dose was comprised of fibers ~3 μm in diameter, 72 percent of which were 10-60 μm long. This was a single dose study with fiber

concentration at 20 mg/m^3 ($\approx$ 40 fibers/cm^3). The rats were sacrificed after 4, 8, 12, and 16 weeks of exposure period, and after 35 and 80 weeks of recovery. No fibers were found in the lung tissue. Histopathological evaluation did not reveal any fibrosis effects or any changes in pulmonary function response.

Warheit [44] conducted a multidose study in rats which were exposed to respirable (1-4 μm diameter), experimental pitch carbon fibers for 1-5 days at concentrations of 50-90 fibers/cm^3 of air. This study was designed to assess the inhalation hazards of pitch carbon fibers in work environment and used a short-term bioassay technique developed by the same author [39]. The effects in terms of histopathological and pulmonary changes were compared with control rats exposed to experimental PAN-based carbon fibers with an average diameter of 4.4 μm. The respirable carbon fiber exposure caused only transient, dose-dependent, inflammatory response. The fiber induced inflammation effects were found reversible with in 10 days of the exposure. Consistent with Waritz, [43] there were no significant pulmonary function changes or evidence of fibrosis in either the pitch- or PAN-based carbon fibers. The author concluded that unlike the silica-based asbestos fibers, pitch-based carbon fibers do not cause pulmonary fibrosis.

Based on the studies reviewed here, it appears that toxicity of respirable, raw carbon fibers and composite dust is substantially different from well known carcinogenics such as asbestos and man-made mineral fibers. The toxic hazards of carbon fibers and dust rank significantly lower compared with the crystalline silica dusts. Luchtel [45] has suggested that, "composite dusts should be regarded as more hazardous than the so-called nuisance dusts."

TOXICITY OF COMBUSTION PRODUCTS FROM COMPOSITES

The burning of fiber composites generates heat and combustion products that consist of a complex mixture of gases and visible products of incomplete combustion, collectively referred to as smoke. The nature of these products depends upon the composition of the burning material(s) and the transient thermo-oxidative conditions during the fire. Smoke composition varies significantly with the change in burning conditions and fire growth rate. At any stage of fire development, the smoke stream contains a mixture of evolved gases, vapors, and solid particles. [46-47]

Aerosols constitute the visible component of smoke and are comprise of aggregates of solid particles adsorbed with combustion vapors and gases. Aerosols (including fibers) are classified in terms of the physical nature and size of its components. [48] Airborne particles vary widely in size from submicron to many microns. Smaller particles stay suspended in air longer and due to their greater surface area are more likely to adsorb chemical vapors from the smoke. The physiological effects of human exposure to fire effluent depends upon the size distribution and solubility characteristics of the aerosols, which determine the depth of penetration in the lungs and the degree of absorption inside the body. [15]

The National Aeronautics and Space Administration (NASA) [49] led a government-wide effort to characterize the carbon fibers emitted from combustion of fiber composites used in aircraft parts. The initial focus of this study was to evaluate environmental hazards due to the conductive carbon fibers. [49-50] It was feared that accidental release and dispersal of carbon

fibers from composites longer than 1 mm could cause shorting or arcing in power distribution lines and other electrical equipment. A series of large-scale fire tests with composites were conducted to simulate aircraft fires. One series of tests were conducted by the Naval Weapon Center (NWC) at China Lake, CA, facility with composites suspended above 15.2 m jet fuel (JP-5) pool fires. The NWC tests subjected actual aircraft composite parts, a Boeing 737 spoiler and an F-16 fuselage section, to flames for 4-6 minutes. Collection of fibers released during the fire tests was achieved through adhesive coated papers 20 x 25 cm located on elevated platforms 0.3 m above ground. However, this sampling procedure was found inadequate due to the low amounts of single fibers collected. The massive smoke plume that reached a height of ~1000 m carried the majority of the single fibers away from the test location to distances beyond the instrumentation limit of 2000 m. Sampling size was also reduced due to difficulties in separating the fibers from the paper, thus limiting the number of fibers analyzed for size distribution. [51]

Another series of large-scale tests fire tests were conducted at the U.S. Army's Dugway Proving Ground, UT. [49, 51] Two of these tests were specifically designed to measure the amount of carbon fibers released from burning large amounts of composite parts. In these tests, carbon-epoxy composite parts weighing about 45 kg were burned in 10.7-m-diameter JP-4 jet fuel pool fires for 20 minutes. The fire-released fibers were collected using an array of filters suspended inside the smoke plume at a height of 40 m. The filters consisted of stainless steel canisters with stainless steel mesh to trap the fibers carried in the smoke plume. Complete details of these large-scale tests and the methodology for collecting the fibers have been reported by Bell. [51]

Collected fiber samples from these tests were analyzed for fiber count and size distribution by optical and electron microscopy. The results indicated that fibers are released in several forms ranging from single fibers to hundred-fiber clusters and fragmented pieces of composite laminate. [52] Single fibers constituted less than 1 percent of the carbon fiber mass initially present in the composite. The tests indicated that under certain conditions involving thin composites with turbulence (e.g., air blast or explosion) the total number of single fibers released from burning composite parts increased significantly. Based on the original carbon fiber mass, a threefold increase in the total mass of collected single fibers was noted under turbulent fire conditions. [49, 51] Microscopic analysis of the fiber size distribution revealed an average length of 2-3 mm. An important finding was that over 70 percent of the fibers collected from burn tests were smaller than one millimeter. The proportion of fibers smaller than 1 mm increased to 98 percent in tests involving both fire and explosion. Since only 2 percent of the fibers generated were longer than 1 mm, it was concluded that the release of carbon fibers from an aircraft crash fire does not pose a significant threat to electrical and electronic equipment in the vicinity.

Microscopic fiber analysis revealed that fiber diameter was significantly reduced in the fire due to fiber oxidation and fibrillation. Figure 4 shows the frequency distribution of fiber diameters collected in a burn test versus the original carbon fibers.

The collected fibers had a mean diameter in the range of 4.2 μm versus 7 μm for the virgin fibers. [51] At extreme flame temperatures (>900°C) and under oxygen-rich test conditions, large amounts of fibers were completely consumed through oxidation. The overall fiber diameter is reduced drastically inside the flame after the fibers are released from the composite.

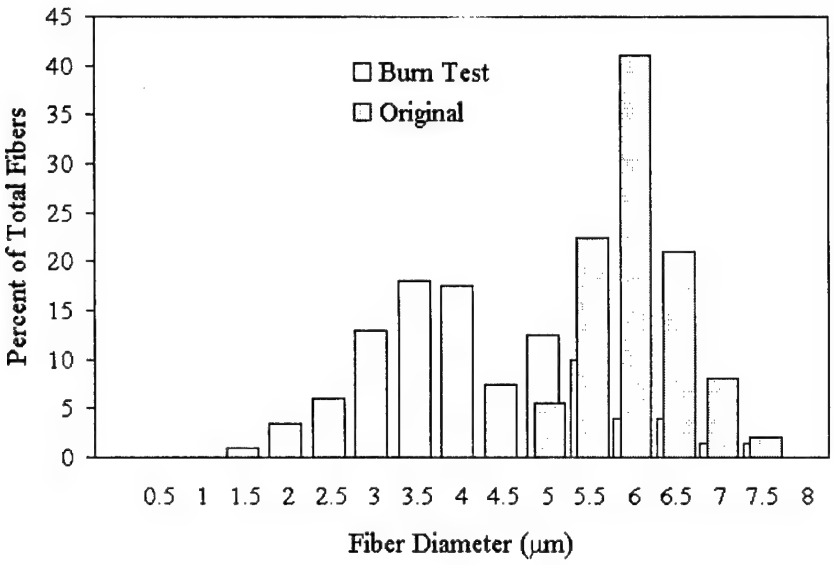

FIGURE 4. COMPARISON OF FIBER DIAMETER DISTRIBUTION OF ORIGINAL CARBON FIBERS WITH THOSE COLLECTED IN THE BURN TEST

Since incineration of fiber composites generates micron-sized fibers that can be inhaled and lead to potential health hazards, further efforts were devoted towards the physical characterization of these fibers using scanning electron microscopy (SEM).

Seibert [7] summarized the results of SEM analysis for respirable fibers (diameter <3 μm, length <80 μm) which constituted less than 24 percent of the total fibers released from burning composites. The respirable fibers had an average diameter of 1.5 μm and were 30 μm long. Overall, the fiber size spectrum ranged from ≈ 0.5 to 5 μm in diameter. Further studies were undertaken to quantify the respirable fiber concentrations and to determine the potential exposure levels. One approach was based on theoretical predictions derived from fiber release data obtained in laboratory and larger-scale jet fuel fire tests using a passive sampling technique. Sussholz [52] estimated that an aircraft fire involving fiber composites would release $5x10^{11}$ respirable fibers per kilogram of the total carbon fibers released (or five percent by weight). This quantity corresponds to an estimated peak exposure of 5 fibers/cm^3 with an upper limit exposure estimated at about 320 fibers/cm^3-s within the smoke plume. This exposure is about 1/10th of the OSHA-permitted [29] maximum exposure level of 3000 fibers/ cm^3 per 8-hour day for asbestos fibers.

A second approach entailed measurement of fiber concentration via direct sampling of fibers from the smoke plume in large-scale tests conducted at Dugway Proving Grounds, as described earlier. All fibers with an L/D ratio greater than 3 were counted and fiber concentrations were determined for the 20-minute burn time. The results indicated an average fiber concentration of less than 0.14 fibers/cm^3. This is ten times lower than the OSHA mandated criteria for asbestos exposure. [29] Lacking evidence of any known pathological effects, the authors concluded that carbon fiber exposure should be treated in the same manner recommended by NIOSH for fibrous glass. [49]

Sussholz [52] conducted an investigation on the size reduction of the carbon fibers in fires. This study attributed reduced diameter fibers to partial surface oxidation and fibrillation effects–splitting of fibers into smaller, finer fibrils due to surface pitting and or surface flaws. Fibrillation of carbon fibers correlated with the presence of metal impurities and morphological flaws such as voids in the graphitic structure. Oxidation effects were observed to be severe in regions of low crystalline density. Elemental analysis of the carbon fibers confirmed the presence of sodium impurities. Using a modified thermogravimetric analysis (TGA) instrument for higher heating rates, Sussholz examined the role of sodium impurities in the oxidation and fibrillation of carbon fibers under nonisothermal conditions. TGA tests on T300 and T600 carbon fibers made with the same precursor but having different levels of sodium indicated that the presence of sodium lowered the oxidation resistance of the fiber. [52] Ismail presented further evidence on the role of sodium in lowering the oxidation resistance of carbon fibers. [53]

The U.S. Coast Guard conducted a series of tests to characterize the graphite fibers emitted from burning graphite/epoxy composites. [54] The laboratory-scale tests were conducted using the cone calorimeter at 50 and 75 kW/m^2 on composite parts used on the HH-65A helicopter. A modified sampling system was used in the cone calorimeter to maximize the collection of fibers after the epoxy resin was completely burned. The fiber size distribution was determined through SEM analysis.

The Coast Guard study indicated that 23 percent (by weight) of the fibers generated were in the respirable range. Overall, the fiber diameter ranged between 0.5-9 μm and the length was between 3-210 μm. The mean fiber diameter and lengths were 2.5 and 52 μm, respectively. In a separate test series, 48- x 48-cm^2 sections of graphite/epoxy composite were subjected to flaming heptane. [54] Analysis of the fiber size distribution indicated that the diameters ranged between 0.5-5.0 μm with a mean diameter of 2.4 μm. The scatter in the fiber lengths was much greater in pool fires data, and ranged between 5-900 μm with a mean fiber length of 77 μm.

Additional data on the characteristics of the airborne fibers have come from recent aircraft postcrash investigations. Mahar [55] measured fiber concentration (fibers/cm^3) and aerodynamic diameter of the fibers collected at a military jet crash site. Fiber samples were collected from the personal respirator filters worn by the investigators fitted with 25-mm cassettes containing 0.8-μm mixed cellulose ester filters. Mahar reported that there is a significant increase in the particulate levels during cleanup operations with the disturbance of the aircraft wreckage. Less than 20 percent of the collected fibers were found respirable with aerodynamic diameter smaller than 10 μm. Microscopic analysis revealed that respirable fibers were approximately 2 μm in diameter and 7-8 μm long. The total concentrations of the fibers collected from breathing air zones ranged from 0.02-0.06 fibers/cm^3. The Navy guidelines limit exposure to a time-weighted average of 3.5 fibers/cm^3 of air and a maximum of 10 fibers/cm^3 over a 40-hour workweek. [55]

To date, no toxicological studies have been conducted to assess the health effects of inhalation of carbon fibers released in fires. No epidemiological or exposure studies could be found on personnel exposure to fibrous particles. Forrest et al. [56] evaluated the toxicity of gaseous products from burning composites material made of PAN-based carbon fiber impregnated with epoxy resin. The carbon fiber had a nominal diameter of 7 μm. Fisher rats were exposed to combustion products for 30 minutes and then observed for 14 days as described in the National

Bureau of Standards (NBS) test. [8] Based on LC_{50} values of the evolved gases, the composite material was determined to be no more toxic than burning wood. However, this study was designed to measure the acute toxicity of only the combustion gases, not the toxicity of carbon fibers that may be carried in the smoke stream.

The Civil Aviation Authority [57] in the United Kingdom investigated the toxic nature of combustion products from composites. Samples of composite materials used in structural components of a public transport jet aircraft and a helicopter were tested in a combustion chamber. Samples were subjected to a flaming heat source at a temperature of 1150°C ± 50°C. The chemical analysis of the combustion products via gas chromatography and mass spectroscopy revealed several organic chemicals. The exact composition of the organic compounds was not presented. Although no fibers were found in the soot filters, the visual inspection of the burnt samples indicated evidence of surface pitting and fiber fibrillation. In a previous study, Sussholz [52] reported similar evidence of surface changes during carbon fiber degradation in a TGA instrument.

US Air Force Toxicology Division conducted a series of tests using a modified University of Pittsburgh apparatus [58] for evaluating combustion toxicity of advanced composite materials used in military aircraft. Preliminary studies focused on the morphology and chemical composition of organic compounds associated with particulates carried in the smoke affluent from burning composites. The test materials consisted of carbon fiber impregnated in a modified bismaleimide resin. [59-61] SEM and digital image processing were used to determine the size distribution of particles. The analyses did not reveal the presence of any fiber-shaped particles (L/D >3). Particles were classified in terms of count median diameter, <1, 1-5, 5-10, and >10 μm. Larcom [59] reported that 40 percent of the particles were of respirable dimension with aerodynamic diameter ≤5 μm. These fibers are small enough to penetrate the tracheobronchial airways. Approximately 15 percent of particles had an aerodynamic diameter ≤1 μm, which can be deposited at the alveolus. The study did not report the fiber length measurements.

Mass spectroscopy of chemicals associated with the particulate matter revealed a large number of chemical species. Lipscomb [60] identified 90 different chemicals which can be broadly classified as polycyclic aromatic hydrocarbons, nitrogen-containing aromatics such as aniline, and phenol-based organic compounds. Several of these chemicals, e.g., aniline, quinoline, and toluidine, are known to induce mutagenic and carcinogenic effects in animals. [60] Further work with rats is underway to study the toxic potential of these chemical agents transported by the particulate matter. [61]

RISK MITIGATION

The health risks associated with carbon fiber exposure are not clearly understood. Although there is a lack of scientific data which prove that the risks of carbon fiber inhalation are similar to silica-based fibers, it is prudent for safety personnel engaged in various stages of an aircraft mishap response to take equivalent precautionary measures. The US Air Force Advanced Composites Program [62-63] has developed guidelines establishing minimum safety and health protection requirements for firefighters, investigators, and cleanup crews in accidents involving advanced composite materials. Firefighters are required to wear self-contained breathing

apparatus, chemical protective clothing, leather gloves, and neoprene coveralls to minimize exposure to fibers. After the fire is extinguished, the debris is sprayed with a fixing agent (e.g., polyacrylic acid) to agglomerate the loose fibers and frayed edges of composite parts. These guidelines have been adopted by the US Navy, the Federal Aviation Administration, and the National Transportation Safety Board for aircraft accident investigation, cleanup, and recovery tasks. The Royal Air Force has developed similar procedures for the mitigation of hazards under these conditions. [64] A handbook from the Association of the Suppliers of Advanced Composite Materials (SACMA) discusses safe handling of composite materials during the manufacturing processes. [65] SACMA considers skin irritation due to mechanical abrasion to be the principal health hazard due to carbon fibers which can be minimized through proper protective wear.

CONCLUSIONS

Structural applications for fiber-reinforced polymer composites in commercial transport aircraft structures have increased significantly over the last decade and will continue to grow in next-generation aircraft. The primary fire hazard of interior and secondary composites used in aircraft cabin and fuselage components is the heat release rate and the toxicity of the gaseous combustion products from the burning polymer matrix. The aircraft cabin occupants are exposed to this hazard during an impact-survivable accident.

The incineration of external structural aircraft components results in hazardous conditions for fire, rescue personnel and investigation and recovery teams in the immediate postcrash situation. Release of a mixture of gaseous, particulate, and fibrous combustion products of unknown composition poses unique protection problems due to lack of proper understanding of the hazards related to composite materials. Adverse health effects in several recent accidents have been reported by firefighters and rescue personnel responding to postcrash aircraft fires involving fibrous composite materials. The reported health effects range from skin and eye irritation to severe respiratory problems. Airport fire fighting and rescue personnel are adopting conservative protection measures in absence of detailed knowledge of the health effects of carbon fibers released in aircraft fires.

Fiber dimensions and deposition in the deep lung determine the toxicity of inhaled fibers. Animal studies have indicated that long, durable fibers that reach the alveoli cause greater pathologic effects than the shorter fibers which are cleared by the alveolar defense mechanism. Evidence of these effects has come from studies on toxicity of silica and asbestos. Respirable carbon fibers are generated by oxidation and fibrillation under extreme thermal conditions (temperatures >1000°C) representative of postcrash jet fuel fires. During crash investigation and recovery operations, fibers become airborne and can penetrate the skin surface of responding personnel. The eroded fibers are small enough to be inhaled and deposited in the deep lung, transporting with them condensed organic chemicals generated in the fire.

Studies on exposure to respirable fractions of raw PAN- and pitch-based carbon fibers do not indicate significant adverse health effects such as pulmonary fibrosis. Other studies involving occupational exposure to carbon fibers and dusts generated during manufacturing of composites do not provide adequate evidence of fibrosis or carcinogenic effects. To date, epidemiological studies have not been conducted to assess human exposure to carbon fibers released from

burning composites nor have animal studies been published on the role of such fibers in pulmonary toxicity. Recent studies have identified a large number of hazardous chemicals that are adsorbed on particulates generated during combustion of advanced composites. Although the exact composition of chemical products is specific to the burning material, the spectrum of organic compounds includes PAHs, nitrogenous aromatics, and phenolics. Several of these chemicals are known mutagens and carcinogens in animals. However, little is known about the toxicological significance of these agents when respired with particulate matter including fibers. Further work is also needed to assess the implications of any synergistic interactions between the various combustion products.

REFERENCES

1. National Research Council, 1996. New Materials for Next-Generation Commercial Transports, Committee on New Materials for Advanced Civil Aircraft, National Materials Advisory Board, NMAB-476, National Academy Press, Washington, D.C.

2. Tannen, K., 1993. "Advanced Composite Materials," *Fire and Arson Investigator*, 1, pp. 50-51.

3. Bickers, C., 1991. "Danger: Toxic Aircraft," *Jane's Defense Weekly*, p. 711.

4. Gaines, M., 1991. "Composites Menace Crash Teams," *Flight International*, p. 17.

5. Anon, 1995. "Danger: Fibers on Fire," *Professional Engineering*, 8 (11), pp. 10-12.

6. Wright, J., August 1997. Personnel Communication, Airport Technology Research and Development, Federal Aviation Administration, Department of Transportation.

7. Seibert, J. F., 1990. Composite Fiber Hazard, Air Force Occupational and Environmental Health Laboratory, Brooks AFB, TX, AFOEHL Report 90-EI00178MGA.

8. Levin, B. C., A. J. Fowell, M. M. Birky, M. Paabo, A. Stolte, and D. Malek, 1982. Further Development of a Test Method for the Assessment of the Acute Inhalation Toxicity of Combustion Products, Center for Fire Research, National Bureau of Standards, Washington, D.C., NBSIR 82-2532.

9. Levin, B. C., V. Babrauskas, R. G. Gann, M. Paabo, R. H. Harris, R. D. Peacock, and S. Yusa, 1991. Toxic Potency Measurement for Fire Hazard Analysis, National Institute of Standards and Technology, Gaithersburg, MD, NIST SP 827.

10. Hall, J. R., 1997. "Whatever Happened to Combustion Toxicity?" *Fire Technology*, Technical Note, First Quarter, pp. 351-371.

11. Purser, D. A., 1995. Toxicity Assessment of Combustion Products, SFPE Handbook of Fire Protection Engineering, Society of Fire Protection Engineers, ed. P. J. DiNenno, Chap. 2-8, pp. 85-146.

12. Hartzell, G. E., 1996. "Overview of Combustion Toxicology," *Toxicology*, 115, pp. 7-23.

13. Speitel, L. C., 1995. Toxicity Assessment of Combustion Gases and Development of a Survival Model, Federal Aviation Administration, FAA Technical Center, DOT/FAA/AR-95/5.

14. Henderson, R. F., 1995. Toxicity of Particulate Matter Associated with Combustion Processes, Fire and Polymers, American Chemical Society Symposium Series, ed. Gordon L. Nelson, Chap. 5, pp. 28-66.

15. Hill, I. R., 1996. "Reactions to Particles in Smoke," *Toxicology*, 115, pp. 119-122.

16. Stover, D., 1991. "Composites Use Increases on New Commercial Transports," *Advanced Composites*, Sept./Oct., pp. 30-38.

17. Federal Aviation Administration, 1997. Handbook: Manufacturing Advanced Composite Components for Airframes, Department of Transportation, DOT/FAA/AR-96/75.

18. Sorathia, U., R. Lyon, T. Ohlemiller, and A. Grenier, 1997. "A Review of Fire Test Methods and Criteria for Composites," *SAMPE Journal*, 33 (4), pp. 23-31.

19. Schmidt, R., 1983. Composite Materials and Aircraft Structure, ISASI Forum, Number 3, pp. 21-27.

20. Warheit, D. B., 1993. Fiber Toxicology, Academy Press, New York.

21. Warheit, D. B., 1995. Contemporary Issues in Fiber Toxicology, *Fundamental Applied Toxicology*, 25 (2), pp. 171-183.

22. Vu, V., 1988. Health Hazard Assessment of Nonasbestos Fibers, Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.

23. International Agency for Research on Cancer (IARC), 1988. IARC Monograph on the Evaluation of Carcinogenic Risks to Humans From Man-Made Mineral Fibers, Lyon, France.

24. Hesterberg, T. W., W. C. Miller, E. E. McConnell, J. Chevalier, J. G. Hadley, D. M. Bernstein, P. Thevenaz, and R. Anderson, 1993. "Chronic Inhalation Toxicity of Size-Separated Glass Fibers in Fischer 344 Rats," *Fundamental and Applied Toxicology*, Vol. 26, pp. 464-476.

25. Lippman, M., 1993. Biophysical Factors Affecting Fiber Toxicity, Fiber Toxicology, ed. D. B. Warheit, Academy Press, New York, pp. 259-303.

26. Lockey, J. E. and N. K. Wiese, 1992. "Health Effects of Synthetic Vitreous Fibers," *Clinics in Chest Medicine*, 13 (2), pp. 329-340.

27. Kennedy, G. L. and D. P. Kelly, 1993. Introduction to Fiber Toxicology, Fiber Toxicology, ed. D. B. Warheit, Academy Press, New York, pp. 15-40.

28. Lehnert, B. E. and G. Oberdorster, 1993. Fate of Fibers in the Lower Respiratory Tract and Fiber-Induced Lung Diseases, Fiber Toxicology, ed. D. B. Warheit, Academy Press, New York, pp. 349-369.

29. OSHA, 1995. General Industry Safety and Health Standard, U.S. Department of Labor, OSHA 3095.

30. NIOSH, 1977. Criteria for a Recommended Standard: Occupational Exposure to Fibrous Glass, Publication No. 77-152.

31. Bishop, E. C. and H. S. Clewell, eds., 1991. Conference on Advanced Composites, March 5-7, San Diego, CA.

32. Kutzman, R. S. and H. J. Clewell, eds., 1989. "Conference on Occupational Health Aspects of Advanced Composite Technology in the Aerospace Industry," *Applied Industrial Hygiene*, Special Issue, December, pp. 1-85.

33. Thomson, S. A., 1989. Toxicology of Carbon Fibers, Proceedings Conference on Occupational Health Aspects of Advanced Composite Technology in the Aerospace Industry, Health Effects and Exposure Considerations, pp. 164-176.

34. Martin, T. R., S. W. Meyer, and D. R. Luchtel, 1989. "An Evaluation of the Toxicity of Carbon Fiber Composites for Lung Cells In Vitro and In Vivo," *Environmental Research*, 49 (2), pp. 246-261.

35. OSHA, 1992. Occupational Health and Safety Administration, Department of Labor, Final Rule: Occupational Exposure to 4, 4' Methylenedianiline (MDA) for 29 CFR parts 1910 and 1926, Federal Register 57 (154): 35639.

36. OSHA, 1992. Occupational Health and Safety Administration, Department of Labor, Polymer Matrix Materials: Advanced Composites, Technical Publication, pp. 12-13.

37. Vu, V., J. C. Barrett, J. Roycroft, L. Schuman, D. Dankovic, P. Baron, T. Martonen, W. Pepelko, and D. Lai, 1996. "Chronic Inhalation Toxicity and Carcinogenicity Testing of Respirable Fibrous Particles," *Regulatory Toxicology and Pharmacology: RTP*, 24 (3), pp. 202-212.

38. Henderson, R. F., K. E. Driscoll, J. R. Harkema, R. C. Lindenschmidt, I. Y. Chang, K. R. Maples, and E. B. Barr, 1995. "A Comparison of the Inflammatory Response of the Lung to Inhaled Versus Instilled Particles in F344 Rats," *Fundamental and Applied Toxicology*, 24 (2), pp. 183-197.

39. Warheit, D. B. and M. A. Hartsky, 1997. Initiating the Risk Assessment Process for Inhaled Particulate Materials: Development of Short-Term Inhalation Bioassays, *Journal of Exposure Analysis and Environmental Epidemiology*, 7 (3), pp. 313-325.

40. Kwan, J. K., 1990. Health Hazard Evaluation of the Post-Curing Phase of Graphite Composite Operations at the Lawrence Livermore National Laboratory, Livermore, CA, Ph.D. thesis, UCRL-LR-104684.

41. Owen, P. E., J. R. Glazier, B. Ballantyne, and J. J. Clary, 1986. "Subchronic Inhalation Toxicology of Carbon Fibers," Journal of Occupational Medicine, **28**, pp. 373-376.

42. Thomson, S. A., R. J. Hilaski, R. Wright, and D. Mattie, 1990. Nonrespirability of Carbon Fibers in Rats From Repeated Inhalation Exposure, Chemical Research, Development, and Engineering Center, Aberdeen Proving Ground, MD, AD-A228-196/HDT.

43. Waritz, R. S., C. J. Collins, B. Ballantyne, and J. J. Clary, 1990. "Chronic Inhalation of 3 μm Diameter Carbon Fibers," *The Toxicologist*, 10 (1), p. 70.

44. Warheit, D. B., J. F. Hansen, M. C. Carakostas, and M. A. Hartsky, 1994. "Acute Inhalation Toxicity Studies in Rats with a Respirable-Sized Experimental Carbon Fiber: Pulmonary Biochemical and Cellular Effects," *American Occupational Hygiene*, 38, pp. 769-776.

45. Luchtel, D. L., 1993. Carbon/Graphite Toxicology, Fiber Toxicology, ed. D. B. Warheit, Academy Press, New York, pp. 493-521.

46. Caldwell, D. J., K. J. Kuhlmann, and J. A. Roop, 1995. Smoke Production From Advanced Composite Materials, Fire and Polymers II, American Chemical Society Symposium Series, ed. Gordon L. Nelson, Chap. 24, pp. 366-375.

47. Chaturvedi, A. K. and D. C. Sanders, 1995. Aircraft Fires, Smoke Toxicity, and Survival: An Overview, DOT/FAA/AM-95/8, FAA Civil Aeromedical Institute, Federal Aviation Administration, Washington, DC.

48. Ness, S. A., 1991. Air Monitoring for Toxic Exposures, Van Nostrand Reinhold, New York, pp. 5-13.

49. Office of Science and Technology Policy, 1980. Carbon/Graphite Composite Materials Study, Third Annual Report, Washington, DC.

50. Kalekar, A. S., J. Fiksel, P. K. Raj, and D. B. Rosenfield, 1979. An Assessment of the Risks Presented by the Use of Carbon Fiber Composites in Commercial Aviation, NASA Contract Report, NAS1-1538.

51. Bell, V. L., 1980. Potential Release of Fibers from Burning Carbon Composites, NASA N80-29431.

52. Sussholz, B., 1980. Evaluation of Micron Size Carbon Fibers Released From Burning Graphite Composites, NASA CR-159217.

53. Ismail, M. K. I., 1991. "On the Reactivity, Structure, and Porosity of Carbon Fibers and Fabrics," *Carbon*, 29 (6), pp. 777-792.

54. Clougherty, E., J. Gerren, J. Greene, D. Haagensen, and R. G. Zalosh, 1997. Graphite Fiber Emissions From Burning Composite Helicopter Components, Draft Report, United Sates Coast Guard, Department of Transportation.

55. Mahar, S., 1990. "Particulate Exposures From the Investigation and Remediation of a Crash Site of an Aircraft Containing Carbon Composites," *American Industrial Hygiene Association Journal*," 51, pp. 459-65.

56. Forrest, V. J., D. L. Geiger, J. H. Grabau, D. B. Curliss, and D. R. Tocco, 1991. Combustion Toxicity of Carbon Composites, Conference on Advanced Composites, San Diego, CA.

57. Greene, G., 1997. Post-Crash Fire Hazards Research, Conference Proceedings, Aircraft Fire Safety, Advisory Group for Aerospace Research and Development, AGARD-CP-587, Germany.

58. Courson, D. L., C. D. Flemming, K. J. Kuhlmann, J. W. Lane, J. H. Grabau, J. M. Cline, C. R. Miller, B. J. Larcom, and J. C. Lipscomb, 1996. Smoke Production and Thermal Decomposition Products From Advanced Composite Materials, US Air Force Armstrong Laboratory, Technical Report AL/OE-TR-1996-0124.

59. Larcom, B. J., J. M. Cline, L. D. Harvey, and D. L. Courson, 1997. "Potential Hazard Associated With Combustion of Advanced Composite Materials," *Military Medicine*, in print.

60. Lipscomb, J. C., K. J. Kuhlmann, J.M. Cline, B. J. Larcom, R. D. Peterson, and D. L. Courson, 1997. "Combustion Products from Advanced Composite Materials," *Drug and Chemical Toxicology*, in print.

61. Lipscomb, J. C. Personal Communication, December 1997. Toxicology Division, Armstrong Laboratory, Wright-Patterson AFB, Ohio.

62. Olson, J. M., 1994. Mishap Risk Control for Advanced Aerospace/Composite Materials: Airforce Systems Command, Advanced Composites Program Office, McClellan AFB, CA, AJ554083.

63. Olson, J. M., 1993. Mishap Risk Control Guidelines for Advanced Aerospace Materials: Environmental, Safety, and Health Concerns for Advanced Composites, USAF Advanced Composites Program Office, McClellan AFB, CA.

64. Andrews, J. W. T., Wing Commander, 1997. Post Crash Management: The Royal Air Force Approach, Conference Proceedings, Aircraft Fire Safety, Advisory Group for Aerospace Research and Development, AGARD-CP-587, Germany.

65. SACMA, 1991. Safe Handling of Advanced Composite Materials, Suppliers of Advanced Materials Association, Arlington, VA.

GLOSSARY.

Alveolus: one of the thin-walled saclike terminal dilations of the respiratory bronchioles, alveolar ducts, and alveolar sacs across which gas exchange occurs between the alveolar air and the pulmonary capillaries. (pl. alveoli)

Alveolitis: inflammation of the alveoli.

Alveolar Macrophage: vigorously phagocytic macrophage on the epithelial surface of lung alveoli where it ingests inhaled particulate matter.

Asbestosis: a disease that is caused by the inhalation of minute asbestos fibers composed of calcium and magnesium silicate. The fibers cause scarring of the tissues in lung and the pleura, the membrane that covers the lungs. Asbestosis has also been linked to lung cancer, particularly in smokers, and in malignant tumor of the pleura called mesothelioma.

Bronchioles: one of the six generations of increasing finer subdivisions of the bronchi. Respiratory bronchioles are the smallest bronchioles (diameter 0.5 mm) that connect the terminal bronchioles to the alveolar ducts.

Bronchus: one of the two subdivisions of the trachea (windpipe) serving to convey air to and from the lungs. Trachea divides into two bronchi (pl. bronchus).

Cytotoxic: detrimental or destructive to the cells.

Epithelium: the purely cellular layer covering all free surfaces, cutaneous, mucous, and serous. (pl. epithelia)

Fibrillation: splitting of a fiber into smaller and or finer fibers due to surface erosion. Erosion can be caused by physical and/or thermal impact or due to dissolution by the action of a chemical.

Fibrosis: formation of fibrous tissue as a reactive process, as opposed to formation of fibrous tissue as a normal constituent of an organ or tissue.

Histamine: a chemical present in body tissue, when released it stimulates production of gastric juices for digestion, also an active agent of allergic reactions that may lead to respiratory problems such as asthma. This condition results due to the constriction of bronchial capillaries that cause a fall in blood pressure.

Histology: study of minute structure of cells and tissues in relation to their function.

Histopathology: study of dealing with cytologic and histologic structure of abnormal or diseased tissue. (Syn. pathologic histology)

Interstitial: relating to spaces or interstices in any tissue, organ, or structure.

Lavage: the washing out of a hollow cavity or organ by copious injections and rejections of fluid.

Macrophages: Mononucleated cells (phagocyte) that are largely scavengers ingesting dead tissue and degenerated cells.

Mesothelioma: a rare neoplasm derived from the lining cells of the pleura and peritoneum which grows as a thick sheet covering the viscera and is composed of fibrous tissue which may enclose glandlike spaces lined by cuboidal cells.

Mesothelium: a single layer of flattened cells forming an epithelium that lines the serous cavities, e.g., pleura, peritoneum.

Mutation: a change in the chemistry of a gene perpetuated in subsequent division of the cells in which it occurs.

Mutagenic: promoting mutation of genes.

Neoplasm: a growth tissue due to abnormal cellular proliferation forming a distinct mass of tissue which may be benign (benign tumor) or malignant (cancer).

Peritoneum: the serous sac consisting of mesothelium and a thin layer of irregular connective tissue that lines the abdominal cavity.

Peritoneal: relating to peritoneum.

Phagocyte: a cell possessing the property of ingesting bacteria, dead cells, or foreign particles.

Phagocytic: relating to phagocytes.

Phagocytosis: The process of ingesting by cells of solid substances, e.g., cells, dead tissue, bacteria, and foreign particles.

Pitch: a residual petroleum product used in the manufacture of certain carbon fibers.

Pleura: the serous membrane enveloping the lungs and lining the walls of the pleural cavity.

Polyacrylonitrile (PAN): a product used as a base material in the manufacture of certain carbon fibers.

Pulmonary fibrosis: a condition in which scar tissue forms in the connective tissues that support the alveoli in the lungs. Scarring can be a reaction to a large number of diseases and conditions that lead to acute to chronic inflammation of lung tissues. (syn. interstitial pulmonary fibrosis)